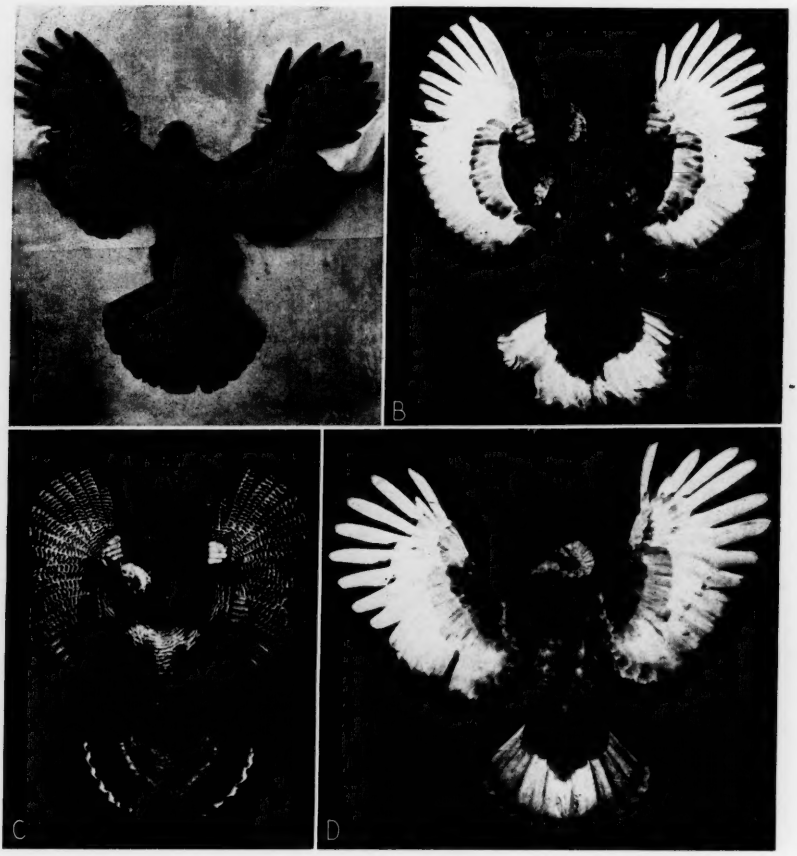
The Journal of HEREDITY



FIVE COLOR GENES AND HOW THEY INTERACT
(SEE PAGE 246)

Eye-Mosaics in Drosophila

Ear Pits and Albinism

A Dwarf Mutation

VOLUME 34 AUGUST, 1943 NUMBER 8

The American Genetic Association

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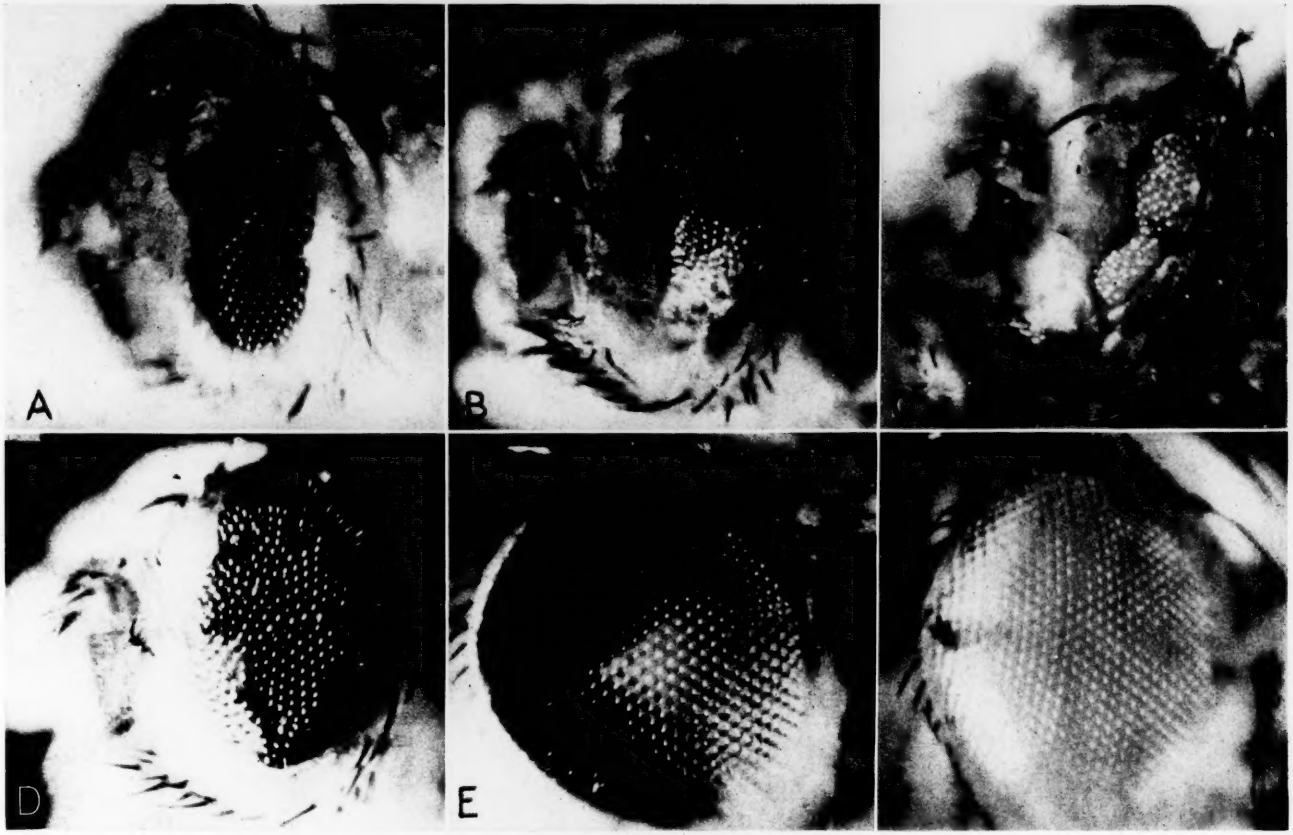

BAR-EYED MOSAICS IN DROSOPHILA

Figure 1

These mosaics are due to crossing over and somatic segregation of chromosomes differing in color factors, during the development of the eye. In the upper series (*A-C*) the mosaic bar-eye grows smaller as the white area increases. In the lower series (*D-F*) the size of the mosaic eye increases as the white area increases. This reversal of growth trends is explained by the fact that in the first series (*A-C*) the bar factor is on the same *X*-chromosome as the white-eye gene (*w*). In the other series (*D-F*) the ultra-bar factor (*Bi*) is linked with the wild-type (red) eye gene, so that the bar character is expressed only in the red areas. For further details see Figure 2.

BAR-EYED MOSAICS IN DROSOPHILA

F. DEMARINIS

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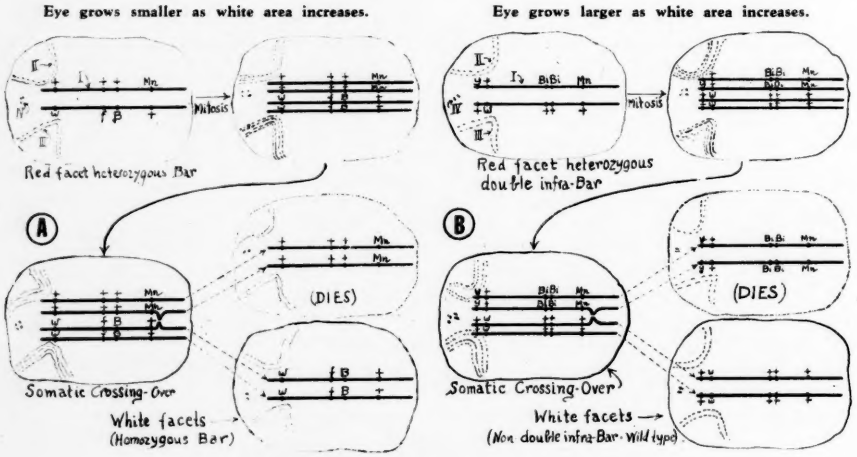
MOSAICISM in organisms has been arousing the interest of biologists for the past thirty years. Not until 1932 when Sturtevant[2] suggested that mosaics might be used to study the developmental effects of genes, did the phenomenon become a really useful research tool. Hersh[1] applied this method to an analysis of the bar-eyed mosaics of *Drosophila melanogaster*. He found that when the sizes of the white areas of these mosaic eyes were plotted logarithmatically against the respective total eye areas, the resulting curve conformed to the relative growth equation, $y = bx^k$. This was the first time that the developmental effects of genes were quantitatively studied by the use of mosaics.

This method in general involves the use of *Drosophila melanogaster* with a specific constitution with respect to the genes being analysed. Such flies are obtained by crossing parents of known genotype, to give the desired gene-constitution in the progeny. For example, in the experiment reported here, mosaics of the bar series were desired which would have the following genetic constitutions: *Mn/wfBB*, *Mn/wfB*, *Mn/wfBiBi* and *yBibiMn/w*. These were obtained in the progeny of four crosses: *Mn/+* ♀ × *wfBB* ♂, *Mn/+* ♀ × *wfB* ♂, *Mn/+* ♀ ×*wfBiBi* ♂ and *yBiBiMn/yBibi* ♀ × *w* ♂. The factor *Mn* was introduced to increase the frequency of mosaics, while the factor *w* is added to distinguish the bar from the non-bar tissue in the mosaic eye. (See Figure 2.)

The eyes of the female progeny were inspected for mosaic condition. Mosaics occur in approximately one out of every 150 cases. The frequency with which they occur depends upon the temperature and upon the nature of the secondary genetic factors.

When, during the development of these flies a genetic change in eye-genes occurs in one of the cells of the optic disc, the adult compound eye will be mosaic. That is, an area of the eye will contain a type of tissue different from the rest, which will be indicative of the genetic change. For example, a fly with *yBiBiMn/w* genetic constitution on completion of its development would normally have a heterozygous double infrabar red eye. If somatic segregation occurred in one of the cells of the optic disc this change would give rise to two new types of cells, *yBiBiMn/yBiBiMn* and *w/w*. The former type of cell presumably dies, while the latter proceeds to devlop as tissue homozygous for white and non-bar. The result of this change is that the adult eye will be red with a patch of white facets. The relative sizes of the red and white areas depend upon the time at which somatic segregation occurs. In proportion as the event occurs earlier in the development of the optic disc, the white area will be larger and the red area will be smaller. The sizes of these areas are measured by counting their respective facets.

When the sizes of the white areas are plotted against the total mosaic eye (red and white) the points conform approximately to the relative growth equation $y = bx^k$, where y equals the number of white facets, x equals the total red and white facets and b and k are constants. In mosaics obtained from crosses in which the bar factor comes from the paternal side, the slope of the line k has a negative value, while when the bar factor comes from the maternal side the value of k is positive. In other words, as the white area increases the total mosaic eye decreases. In the second case, on the contrary, as the white area increases the total eye increases. The photographs included in this report may help to illustrate this point more clearly. Figure 1 *A*, *B*, and *C* are photographs of mosaic eyes of the group *Mn/wfB* in which the bar



REVERSE GROWTH-TRENDS EXPLAINED

Figure 2

A shows the production of mosaic eyes of the type illustrated photographically in Figure 1 *A-C*. Here an increase in number of white facets results in a *decrease* in the size of the eye because the white facets are homozygous for the bar (*B*) gene. This comes about through somatic crossing over in the *X*-chromosome in a fly heterozygous for *B*, whereby the two "Bar" chromosomes find themselves in one daughter cell and the two wild type *X*-chromosomes in the other daughter cell. The latter cell dies because of the lethal effect of the *Mn* gene when homozygous. The situation wherein the increase in the amount of white area results in an increase in the size of the eye (Figure 1 *D-F*) as illustrated in diagram *B*. Here somatic crossing over results in the "double" ultra-bar locus being carried with the lethal combination so that the white facet area is wild type, or more strictly non-double-ultra-bar.

factor comes from the paternal side. Here we see that the eye gets smaller as the white area increases. Figure 1*D, E,* and *F* are mosaic eyes of the *yBiBiMn/w* series in which the bar factor comes from the maternal side. In this group the eye becomes larger as the white area increases. This confirms the prediction made at the start of the experiment that mosaics resulting from a somatic crossing over at some point at the right of the *Minute-n* locus in one case would produce a larger eye and in the other a smaller eye. A detailed discussion of these mosaics from the point of view of the relative growth equation has already been taken up in another paper (*Growth*, March 1943).

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1. Hersh, A. H. *Science*. 80:547. 1934.
2. Sturtevant, A. H. *Proc. Sixth Int. Congress Genetics*, I, 304-307. 1932.

AN ACHONDROPLASIC MUTATION AND THE NATURE OF ITS INHERITANCE

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EARLY Utah families furnish exceptional data for the study of human genetics. The families are large and in cases where polygamy was practiced more than one family per father is involved. This is particularly helpful in tracing the origin of hereditary traits. These families are very much interested in genealogy and in many instances have accurate family records for many generations back, which are invaluable in any study of genetics. The present report of a six-generation pedigree where four generations back achondroplasia appeared as a mutation is based on this excellent material.

Review of Literature

Descriptions of dwarfed individuals have appeared since very early times. Dr. Charles E Snow[11] reported that achondroplasia existed among prehistoric American Indians. He found two Indian skeletons at Moundville, Alabama, which show achondroplasia. The male was between 4 feet 4 inches and 4 feet 6 inches in height and the female 4 feet 1 inch. According to Jenkinson and Kinzer,[7] Parrot described the condition in 1878 and was the first to apply to it the present name.

Morch[8] gives the typical symptoms of achondroplasia as follows: "Dwarf growth, (for men about 130 cm., women about 120 cm.), large head, prominent frontal eminence, root of nose sunken, high palate, short extremities (especially shortening of the humerus and femur), arms so short that the tips of the fingers only reach the femoral trochanters or a little over, 'Main en trident' (short fingers all about the same length, divergence between the 3rd and 4th fingers, short broad hand), defective extension of the elbow joint, very bowed legs, waddling gait, short broad feet, straight vertebral column, and highly pronounced lumbar lordosis."

In 1912 Reschbieth[9] and Barington,[9] in a study of achondroplasia, reported pedigrees of 80 individuals. Of these, 20 showed a family history of the defect in two or more generations. Since 70 cases in a total of 126 cases were females, the writers postulated a sex-difference in susceptibility to the trait.

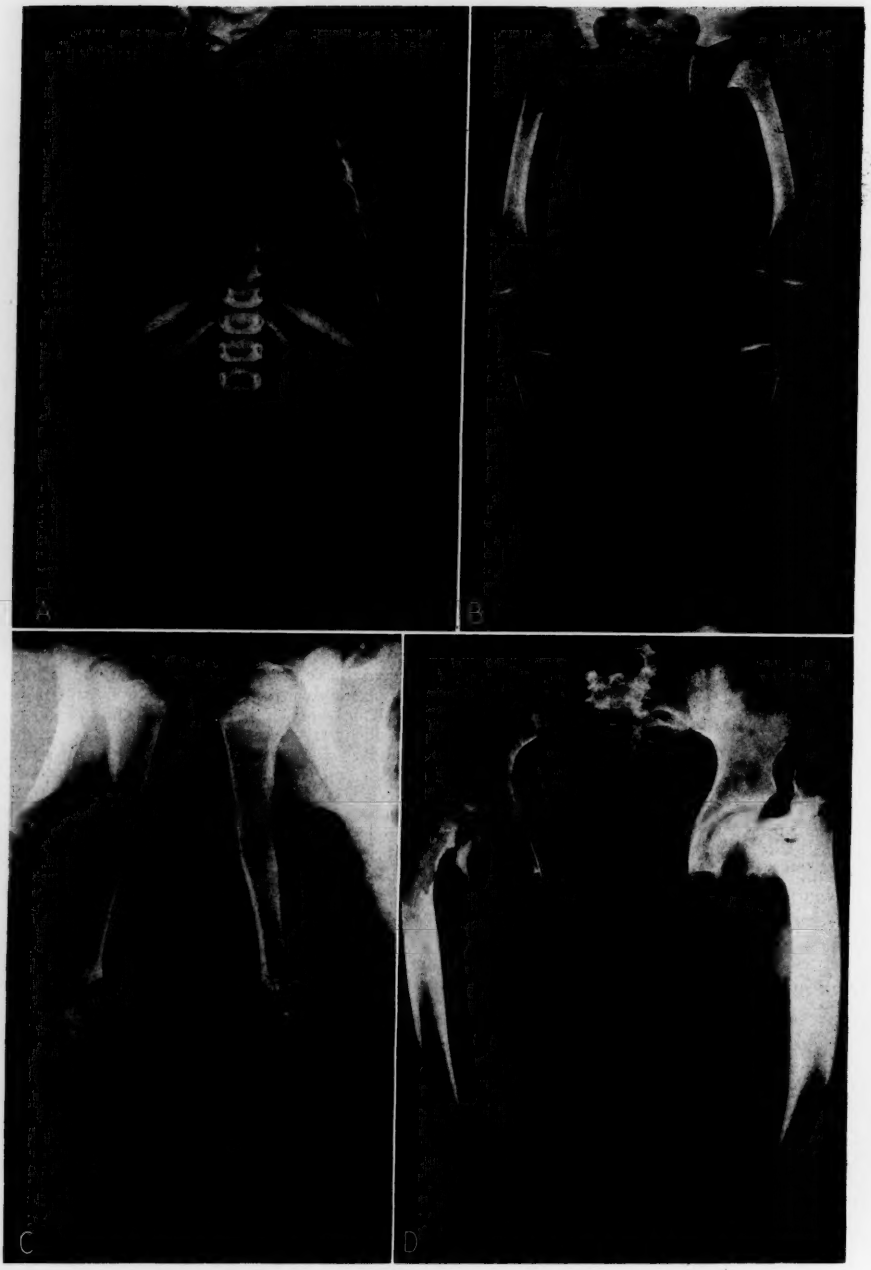
After citing examples reported in the *Treasury of Human Inheritance*, where the achondroplasic character had skipped generations, Davenport[6] stated: "This skipping of generations (of which there are not many cases) would speak against the view that the achondroplastic dwarfism is a simple dominant trait. Also, in most of the pedigrees the achondroplasic dwarf appears as the only case in the family. Perhaps we may conclude with Plate (1913, pp. 349-353) that 'In achondroplasia there is a dominant (growth inhibiting) factor, but that its full expression is often interfered with by other growth-stimulating, or by cancelling or antagonistic factors.'"

Bangson[1] reported the occurrence of achondroplasia in four generations of a family. Two of these cases were produced by normal parents. He draws no conclusions as to the nature of inheritance of the trait, but includes a picture which shows the individual to have short legs and short arms. It is difficult to harmonize this with simple dominant inheritance.

Capinpin[4] reported three successive generations of dwarfism wherein the body was of normal size and the extremities short. He concurred in Davenport's conclusions, that all forms of dwarfing including ateliosis and achondroplasia are governed by multiple dominant inhibiting factors.

Morch[8] reported checking on cases previously reported by Kristine Bonnevie showing recessive inheritance and found them not to be real cases of achondroplasia. In a preliminary report of his own unfinished investigations in Denmark and Sweden, he concluded that

*In the present study of achondroplasia, the writer wishes to express his gratitude for the very generous and helpful cooperation of members of the family studied and sincerely hopes that this paper will prove of interest and value to them. He also wishes to thank Dr. James P. Kerby who very generously took the X-ray pictures and made the diagnosis of each.



BONE DEVELOPMENT OF ACHONDROPLASIC CHILD AND ADULT

Figure 3

The upper two photographs (*A-B*) show x-ray pictures of the torso and legs of a typical achondroplasic child three years of age. Note the extreme shortness of arm and leg bones as compared with the normal. In the normal individual the elbow joint extends nearly to the ilium. (*C-D*) shows arms and leg bones of nineteen year old achondroplasic whose legs were not bowed as is usually the case. The extreme shortness of femur and tibia is again notable.

"Achondroplasia is always hereditary and is inherited dominantly and that it is relatively frequently produced by mutation." He also reported that he found only four cases in Denmark where several cases of achondroplasia occur in the same family and that these occurred only in two generations.

Cockayne[5] states that when sufficient data have been collected, it is probable that achondroplasia will be inherited as are other cases of double dominants. Snyder[10] finds that achondroplasia "is apparently dependent upon the simultaneous presence of two autosomal dominants."

Stockard[3,4] reported achondroplasia of the extremities in several of the domestic breeds of dogs from widely separated countries. He found it to be inherited as a simple Mendelian dominant. In his crosses, the factor produced different degrees of severity of the character depending upon the bone constitution of the breed concerned.

Achondroplasia

In studies on the nature of the inheritance of achondroplasia, writers have not always given adequate descriptions of the defect. It is possible, therefore, that all reported cases of achondroplasia are not identical. According to Bauer, Fischer and Lentz[2] the character varies in intensity in different families. All cases, however, seem to have in common a normal sized body with short legs and arms. The defect reported in this paper appears as follows:

The adult individuals range in height from 53 to 59½ inches. The body is of normal size while the legs and arms are short. The ratio of standing height to sitting height is approximately .6, (height/height sitting in inches for nine individuals being: 58/35, 53.5/30.5, 55/33, 57.5/34.5, 59.5/34.5, 56.7/34.5, 59.5/35.7, 53/34, 34/22). The femur and humerus bones are decidedly short while in the tibia and ulna the shortening is not so marked. The legs in nearly all cases are badly bowed and individuals all show a peculiar waddling gait. The heads are normal in size and shape, and there is no indication of any abnormality of the face, such as pug noses or sunken areas at the base of the nose, as in cases reported by some writers. As a matter of fact, the faces of the affected individuals are quite similar in appearance to the normal members of the family. The hands and feet also seem to be normal. In some families the fingers are quite long rather than short and stubby. There is no indication of any abnormal division of the fingers.

Figure 3 (*A, B*) shows X-rays of a typical three year old achondroplasic child in the family studied. The short arms and bowed legs are evident. The diagnosis as given by the physician who took the pictures is as follows:

Examination of the dorsal and lumbar spine and ribs shows no bony pathology. Each humerus is shorter than the ulna on the corresponding side. There is marked flaring of the upper end of the diaphysis of each humerus. There is considerable spreading of the lower end of the diaphysis of each radius and ulna. Examination of the thighs shows each femur to be shorter and more flaring at each end of the shaft than normal. The epiphyseal line is flared. Each tibia is shorter and thicker than normal and shows marked flaring at the epiphyseal zones. There is a disparity between the length of the humeri and the femora as compared with the ulna and tibia. This is probably some form of congenital chondrodystrophy."

Figure 3 (*C, D*) shows X-rays of an achondroplasic young man 19 years of age of the family studied, whose legs do not show the typical bowed condition. The diagnosis in this case is as follows:

Examination of the pelvis shows presence of two metallic fragments on the right side. Each acetabulum is slightly shallower than normal. There is a partial dislocation of the head of the right femur. There has been a fracture-epiphyseal separation through the epiphyseal line of the neck of the right femur. Shaft of right femur is much smaller than shaft of the left femur. The angle between the neck and shaft of each femur is only about 90 degrees. Examination of the right and left humerus shows them to be shorter and stubbier than normal. Condition is probably some form of congenital chondro-dystrophy.

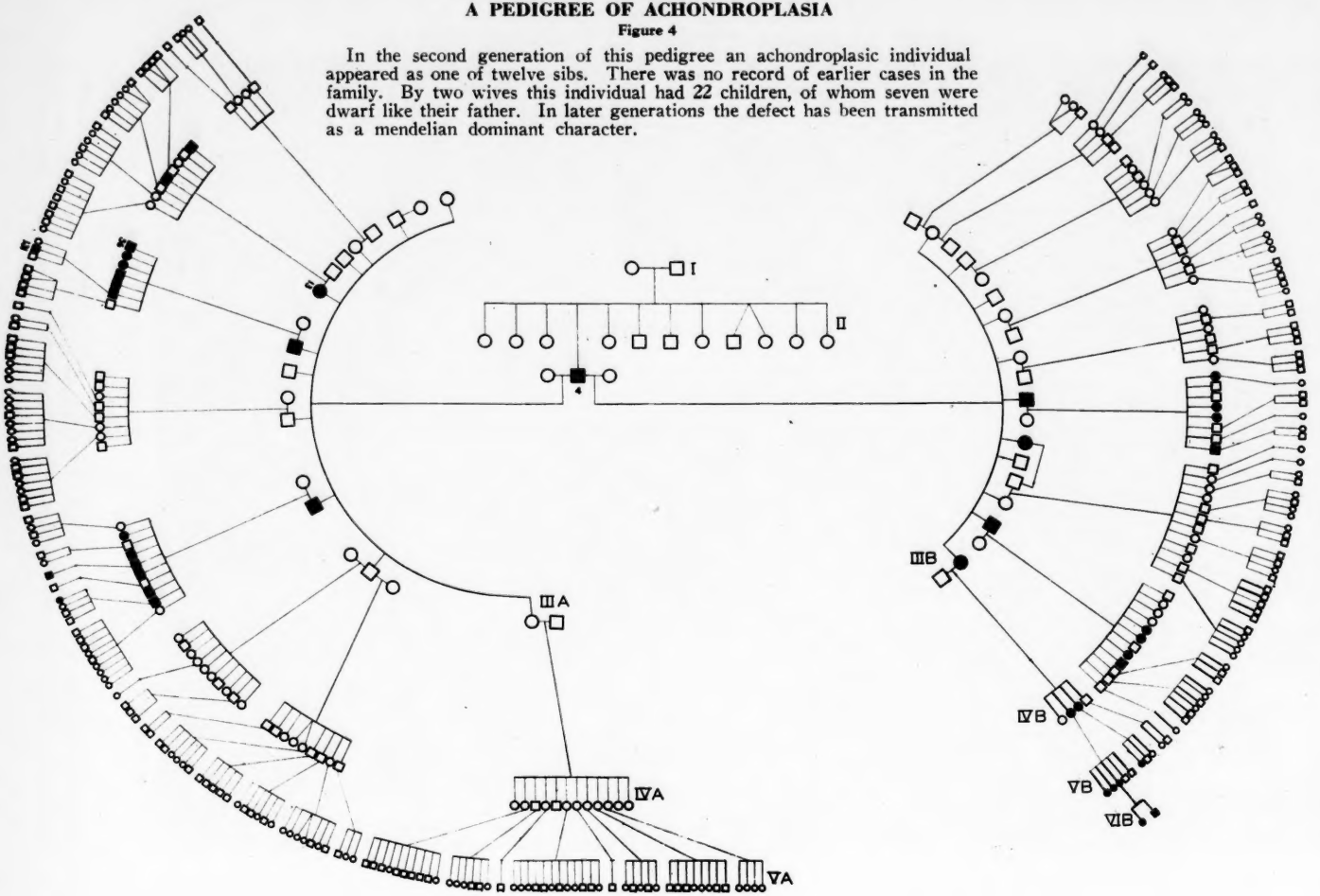
The mentality is in no way affected by the trait. Some of the individuals have been very successful as farmers and sheepmen, and some have held responsible positions in their communities.

When questioned as to when they were first able to detect the trait in their children, two mothers said they were able to tell it at birth. Two others were unable to detect it until the children were nearly a year old, and in one case, older. In most cases when the child began to walk, the legs showed the characteristic

A PEDIGREE OF ACHONDROPLASIA

Figure 4

In the second generation of this pedigree an achondroplasic individual appeared as one of twelve sibs. There was no record of earlier cases in the family. By two wives this individual had 22 children, of whom seven were dwarf like their father. In later generations the defect has been transmitted as a mendelian dominant character.



weakness. They became bowed and inadequate to support the weight of the body. In a few instances where braces have been used at an early age, the legs have remained straight, but are short. Some individuals experience considerable pain if they are on their feet too long. They all, however, seem to be able to do a normal amount of work. In a few cases, the legs are not bowed. No. IV(a) 56 is an example of this type. His mother stated that she was unable to detect the trait in him until he was nine or ten years of age.

A pedigree chart of the family reported is given in Figure 4. It includes the parents and brothers and sisters of II-4, as well as all of his descendants down to and including grandchildren and some great grandchildren. It only includes those great grandchildren coming from one defective parent. This last generation is changing so rapidly that it was thought best not to include the others. The individuals and families are all arranged in order of their age. This chart gives a graphic picture of the origin of the trait and its increase over five generations. The first case of achondroplasia to appear was II-4, the fourth child of a family of twelve. The father and mother and the other eleven children were all normal. Following the appearance of the trait in II-4, it has occurred in some members of each succeeding generation.

II-4 was born in 1833 and died in 1912. He is described by his children as being very short in stature and similar in appearance to his descendants showing achondroplasia. He was an early pioneer in his community and acted as deputy sheriff as well as interpreter between the Whites and Indians. He seemed to be a very industrious and respected citizen. He was a polygamist, having married two women. He had eleven children by each wife. In other cases where the family tree shows more than one wife for a man, the first wife died before the second marriage occurred. The size of families of his children and of later generations was usually large as can be seen from the family pedigree. Eight to twelve children were not uncommon.

Nature of Inheritance

From all available information it would seem that II-4's dwarf stature resulted from a mutation which affected a gene in either the reproductive cell of his father or his mother. No record of the trait occurring in his family before him can be found. His parents were both normal in height as were his eleven brothers and sisters. All other achondroplasic individuals in this study for five generations trace directly back to this man, and in every case, one of the parents shows the trait.

Of the 22 children of II-4, the character appears in seven, occurring in families of both his wives. All seven affected children married, and all but one had offspring, totaling 49, of whom 24 showed the trait. Table I shows the children of II-4, and those of his children and grandchildren, who showed achondroplasia in their families.

TABLE I.—Children in Families Where One Parent Shows Achondroplasia

No. Children In Family	Normal		Achondroplasia	
	Male	Female	Male	Female
11	5	3	2	1
11	3	4	2	2
10	2	2	5	1
7	1	0	4	2
8	3	3	2	0
4	1	1	0	2
12	5	3	1	3
8	4	0	1	3
3	1	1	1	0
1	0	0	1	0
4	2	1	0	1
5	2	0	0	3
3	1	1	0	1
2	1	1	0	0
1	0	1	0	0
2	0	0	1	1
Totals	31	21	20	20

TABLE II.—Distribution of Normals and Achondroplasics

	Observed	Calculated on 1:1 basis	Dev.	P.E.	Dev/P.E.
Normals	52	46	6	3.23	1.86
Achondroplasic	40	46			

Some of the last families on the table are possibly not yet completed. As will be seen from the pedigree chart, defective children occur only in cases where one parent shows the trait. The crosses, therefore, all represent simple Mendelian backcrosses and should produce on the average a 1:1 ratio. Of the 92 children in affected families, 52 are normal and 40 show the defect. The deviation is only 1.86 the probable error and is not significant. The trait occurs equally often in males and in females, and therefore, is undoubtedly a simple autosomal dominant.

The statement that the trait shows complete dominance, however, is only an assumption, since no cases have been reported wherein two affected individuals have married and have had children. Consequently, there is no opportunity to see what the homozygote looks like, or whether it is viable. It might be a more severe form of the defect or even be lethal. Just how the gene produces this defect is not known. It is possible, as suggested by Stockard, that it is through some interference with the normal hormone secretions of the body during development. This interference might produce different results at different stages of development or in different genotypic compositions. This might account for the differences between the bowed legged and the straight legged types. According to Stockard, achondroplasic leg length in dogs is more severe when in the homozygous condition. In the case of the creeper fowls, Landauer and Dunn[12] found the homozygous condition to be lethal.

Selective Value of Achondroplasia

The family studied is large and includes the children of the normal as well as of the defective individuals. It is, therefore, of interest to note the rate of increase of this defect. Since 1833, 41 short individuals have occurred in five generations. Of these, three have died. Their ages at the time of death were: 79, 78, and 61 years. The ages of the survivors range from two years to 79 years. The whole family is vigorous and long lived, and the affected individuals are no exception. While the defect is a decided physical handicap, it seems in no way to shorten life, nor to serve as a handicap to reproduction. Where the rate of reproduction of affected individuals is decreased as compared to normals, it is because of social and phychological attitudes, rather than from physical handicaps. Several writers have stated that normal births were impossible by achondroplasic women. In this family, there is no evidence that this is true. One achondroplasic, III (a) 13, who had eight children, stated that she had normal births in each case attended only by a midwife.

The affected children of II-4, with one exception, had large families, but in the next generation, only seven of a total of 24 defective grandchildren married and had children. In some instances, these individuals have foregone marriage deliberately because they felt that the defect should not be perpetuated. Some have stated that they would not bring children into the world to suffer the embarrassment and handicaps they have experienced. There is also evidence that in some cases, where the normal children of defective individuals have married but have had no children, it has been because of fear that the defect might reappear.

The trait is definitely objectionable and a handicap in selecting a husband or wife. If it is perpetuated for sufficient generations one might expect a tendency for the physical and even the mental standards of the families to be lowered because of the selection against the defect by normal individuals.

Wherever this defective gene is present, it expresses itself in a definite phenotype. It will not be carried over from one generation to the next by normal individuals. An examination of the family chart shows a very definite decrease in the later generations in the rate of reproduction of defectives as compared with the normals. It must be remembered, however, that in many instances, those showing the trait do not marry until late in life. Of the 41 achondro-

plasics born in this family, 19 have married, 15 have children, and 22 are still unmarried.

Can the Trait Be Eliminated?

Since the character is due to the presence of a simple dominant gene, it can be eliminated from the family completely if those people showing the defect fail to reproduce. This might be done by failure to marry, by sterilization, or by practicing some adequate form of birth control. Achondroplasia of the type here reported can only be produced if one or both parents suffer from the defect, or if a new mutation occurs. Normal parents need have no fear of producing affected children even if they themselves come from achondroplasic families. Mutations are rare and it is doubtful if they are any more apt to occur in this family than in another.

Summary

A pedigree is presented showing a mutation to achondroplasia four generations back in a Mormon family. The mutation has been transmitted as a typical Mendelian dominant since its appearance. Distribution of the trait in the population and possibility of eliminating it are discussed.

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THE QUEST FOR A COMMON DENOMINATOR

We all love to generalize. Science itself is a quest for generalizations; yet the broader the generalization the less likely is it to be entirely true. "The task of science is to find and describe such order as exists in a universe in which probably every event has some unique aspects." Enthusiasm for beautiful generalizations can easily lead to impatience with and neglect of the stubborn little facts which refuse to fit. And then there is the ancient epigram: "All generalizations are false, — including this one!"

All of this is by way of calling attention to a recent symposium* on how far there are any general or "fundamental" principles concerning both biological and social studies and governing the relation of individuals to each other and to the groups of which they are members. The occasion was the semi-centennial of the University of Chicago. The authors are twelve professors of philosophy, biology and sociology, with an editor to write the introduction and to emphasize (rather desperately it seemed) the essential unity of the problem as a whole. The following comments may indicate the variety of topics and viewpoints.

The essential difference between a protozoan colony and a multicellular individual is that the latter has established an axis; — i.e. has become polarized. As the parts of a multicellular individual become increasingly differentiated, the need for communication from region to region becomes more acute. Both electrical and hormonal means of doing this have been evolved and both are used to varying degrees in the same organism.

**Levels of Integration in Biological and Social Systems.* Edited by Robert Redfield. 240 pp. The Jacques Cattell Press. 1942.

The transmissive mechanism and integrative technique become increasingly important as the parts become more specialized. "Men in a group must all be alike to the extent, for example, that they can communicate in a mutually understood language; the problem is to keep them sufficiently different so that they have something unique and worthwhile to communicate to one another!" The existence of differences between individuals is the *sine qua non* for organization. "Without functional differentiation there is no social *organization,* but only *aggregation.*" (Jennings). Many of the phenomena of regeneration demonstrate the reality and extent of *organization* over and above what can be seen from the chemistry and anatomical structure of the constituent parts, each considered by itself.

Among all organisms bacteria are most nearly known only by their population characteristics. Those vary, often in unexpected ways, in mixed cultures when the proportions of the various ingredients are changed. Much of what we know about how frogs or cattle behave was learned by studying the behavior of a frog or a cow. But little is known about what a bacterium does or how it behaves although much is known about how *populations* of many kinds of bacteria behave. The cooperative as well as the antagonistic interactions of mixed cultures or enzymatic collaboration ("synergism") will suggest many parallels and analogies with plant and animal ecology.

Allee, in a chapter especially noteworthy for the large proportion of fact and terse summary, tells about social dominance and subordination among vertebrates. This gives a brief glimpse of pertinent points from the extensive recent studies at Chicago on peck-dominance, territoriality, and kindred manifestations of social structures in mammalian and avian populations. The peck-right among hens is not always a simple straight-line hierarchy, although it is often nearly so. It may be related to territoriality, is influenced by heredity, by the physiological state of the individual, and by the memory of past experience. "There is much social inertia in these flocks of hens; — high social rank once won, tends to be held for a long time." Experience of mice with victory or defeat tends to condition them to expect and to gain the same in the future, but it is more easy to condition them downwards then upward. "Success tends to make for continued success, and failure tends to produce continued failure." "—the one in front is at times merely the fastest bird in the flock. So far as true leadership is concerned, it is merely following along ahead of the main flock!" The field observations on variation in the number of offspring per male among sage grouse in Wyoming suggest how very much smaller what Wright calls the "effective number" of males may be than the actual or census number.

Emerson holds that the social structure of insect societies must be perpetuated genically, while man can propagate his social institutions by educating his young. The picture of predestination he presents for the insect societies is extremely Calvinistic in its hues. But man is much freer to work out his own salvation, — at least if his educational system is elastic enough to let him make new trials (which usually turn out to be errors!) now and then.

In societies of monkeys and apes there are wide differences from one species to another in social organization. One having read Carpenter's chapter with its many specific examples, will thenceforth be more skeptical about the validity of conclusions about human social problems based on facile premises about what "primitive man" did. Maybe he and she didn't! Kroeber in the chapter on the societies of primitive man emphasizes the various forms which the idea of kinship has taken, the meaning of clans, whether patrilinear or matrilinear, marriage taboos, etc. But "primitive man" is such an alluring catchall for our private hopes and fears and compulsions that he is not likely to disappear, in all his varied postulated behavior, from social phantasising!

Perhaps the most provocative chapter is the last one. Robert E. Park in "Modern Society" stresses that in tribal times human societies were based largely on the blood tie; i.e. on kinship. But now this partially genetic basis has been in considerable part displaced by such other things as geographical proximity (independent of racial likeness or unlikeness), community of economic or military interests, etc. With the broad outlines of this general conclusion few geneticists would find fault, although some of the detailed remarks and prophecies may inspire indignant denial and hot rebuttal. The biological side of the presentation hardly reaches the clarity of Fisher's chapters on Man and Society a dozen years ago in his *The Genetical Basis of Natural Selection.* Nor is it as lucid as Haldane's comments that natural selection in favor of genetic factors for heroism (i.e. genes which would make their possessor more ready to sacrifice himself for the good of his community or group) can occur only in very small societies knit together by a blood tie.

Although willing to *hope* that Mr. Park is right in the following quotation from page 217, the reviewer, as a parent who has recently been surprised at some of the things in high school and college freshman history texts, is inclined to fear that the metamorphosis hinted in the last sentence has already gone farther than Mr. Parks thinks. "History at any rate does not ask what things are. It has no taxonomic problems because it does not ordinarily seek to make general statements. It merely uses them to elucidate its statements of historical fact. If and when historians seek to make their statements general rather than specific, history ceases to be history and becomes, possibly, sociology."

That groups can show properties and actions which are distinct from the properties of the individual units which compose them few would deny. The existence of as complex a subject as hydrodynamics seems convincing proof that, even with a thing so simple as molecules of water, the whole story of what *groups* of them are and do is not told in their chemistry and hydrostatics. Geneticists may sometimes exaggerate the importance of differences between individuals, since such differences are the raw material of genetics and it is natural for a man to be enthusiastic about that with which he works. But students of social systems just as naturally focus on the way groups are organized. Since it is usually *easier* (in sociology as well as in theoretical mechanics) to describe group uniform elements (individuals) and to assume that the differences in their relations to each other were forced upon them by the organization process itself, a little wishful thinking often ends in grossly underestimating the magnitude and frequency of innate differences between individuals. Park outlines the field of sociology thus on page 218: "But the things which science classifies change. Crowds under certain circumstances become mobs; religious revivals produce religious sects; social movements are transformed into social institutions. It is with these changes and the processes by which they are brought about that sociology and the social sciences, in so far as they assume the character of natural sciences, are ultimately concerned."

Research nearly always proceeds most fruitfully by using mechanistic working hypotheses, even if the worker does have some personal yearning for more mystical views. It would be hard to state the case for this more vividly than in Park's words on pages 231 to 232: "There is — nothing so thoroughly rational and nothing so completely intelligible as a machine. Once one understands how to take a machine apart and put it together again, there is no longer any mystery about it. The thing stands there, as it were, completely unveiled, with nothing either obscure in its past or problematic about its future to inspire speculation or encourage mysticism, since its behavior is completely predictable. This is, in fact, what is meant by making a thing intelligible; it performs this function by treating things as machines, that is,

things that can be taken apart and put together. Where, as in the case of living organisms, science has been able to take things apart but has not been wholly successful in putting them together again, living creatures and life itself have remained, from the point of view of science, more or less a mystery." And for philosophers who may still rely on finding "the good, the true and the beautiful" by sheer reasoning and logic, or who do not appreciate the pragmatic necessity for "test runs" and "pilot plants," there is this frank naming of the most useful touchstone by which scientific truth is distinguished from error: "The successful application of science is the final test of the validity of its findings."

As an integrated organism itself this symposium has something of a split personality. In parts it is a philosophical (at times almost metaphysical) statement of what in 1941 was thought to be the central unity or general truths concerning integration throughout the whole scale of biological and social systems. Whether these parts are worth the reading is largely a matter of one's thirst for this kind of mental diversion. Other parts are factual summaries of what is now known in various fields concerning how individuals behave toward each other when they become organized as groups; and how such groups as wholes may act or be otherwise than might have been expected from the properties or behavior of the individuals which compose them. These summaries do help to bring specialists from other fields up to date. They will be useful initial guides to any who wish to explore further the details of a particular field.

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ON THE PRODUCTION OF POLYPLOIDY IN ALLIUM WITH PARADICHLORBENZENE

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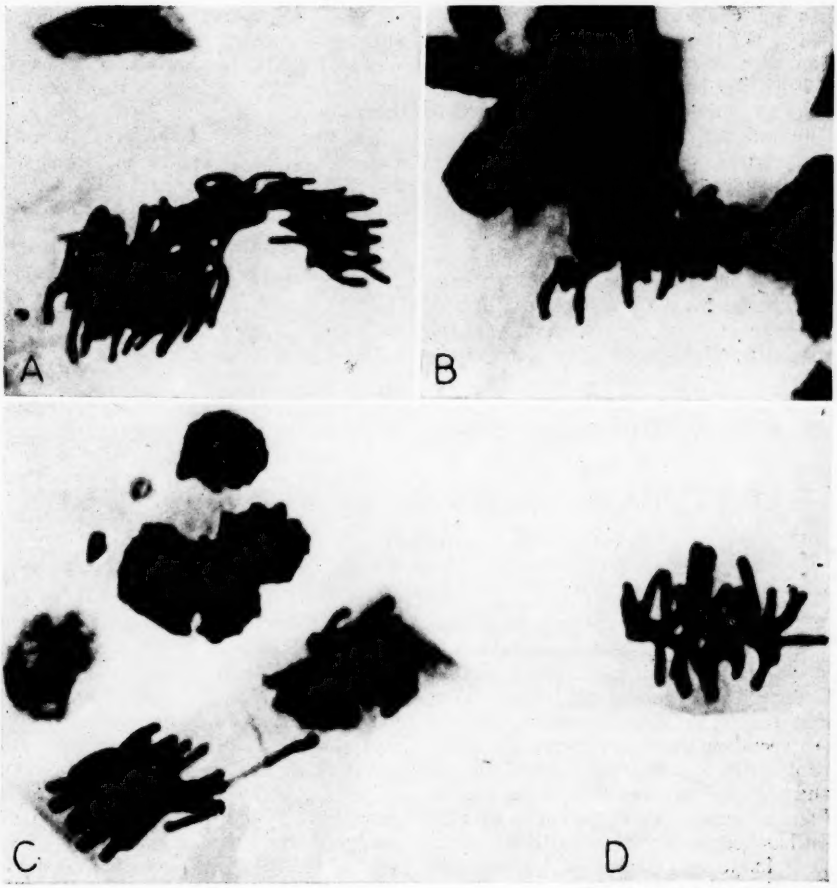
THE work of Levan,[3] Blakeslee and Avery,[1] and Nebel and Ruttle[4] on the production of polyploidy with colchicine led to numerous investigations on the artificial production of polyploidy. Since the discovery of the efficacy of colchicine in the production of heteroploidy, numerous other chemicals have been used with success.

In October, 1940, attempts to control the growth of molds affecting buckwheat seedlings were made with paradichlorbenzene. It was noticed that there was a stunting and thickening of the roots. This led to an investigation of the possibility of increasing the chromosome number with this substance. Search for previous articles on the use of this chemical disclosed the fact that Simonet and Guinochet[5] had successfully employed this substance in producing heteroploidy in *Linum usitatissimum*.

The results reported here were obtained in *Allium* roots taken from seedlings grown on moist filter paper in Petri dishes. Twenty-five seeds were scattered on moist filter paper in each of six Petri dishes. Paradichlorbenzene was weighed and placed on watch glasses in the Petri dishes. The concentrations of the chemical were as follows: 1/20 g., 1/10 g., 1/4 g., 1/2 g., 1.5 g. The experiment was run in duplicate and controls were run at the same time. The seedlings were exposed to paradichlorbenzene for five days at room temperature. At the end of this time, roots were fixed, smeared and examined for mitosis and chromosome number. Roots taken from Petri dishes which contained: 1/4 g., 1/2 g. and 1.5 g. showed no signs of mitosis.

In another experiment, seedlings were allowed to develop until the fourth day in Petri dishes containing only moist filter paper. At the end of this time, they



Photomicrographs by Leo C. Massopust.

NUCLEI OF ONION ROOT-TIPS TREATED WITH PARADICHLORBENZENE

Figure 5

A-B—Heteroploid nuclei of treated root-tips, showing extra sets of chromosomes resulting from treatment. *C-D*—Two adjoining nuclei showing a heteroploid nucleus (*C*) and adjoining it a diploid nucleus (*D*). Note the lagging chromosomes in the nucleus shown at *C*.

were exposed to the fumes of Paradichlorbenzene from 10:00 A.M. to 2:00 P.M., C.S.T. The same quantities of the chemical were used. Twenty-four hours later, the roots were prepared for microscopic observation according to a smear method used by Heitz.[2] Roots taken from Petri dishes which contained 1/2 g. and 1.5 g. showed polyploidy. Abnormal chromosome numbers were found to be present in the dividing nuclei. Polyploid numbers were especially apparent during the metaphase stages. (Figure 5*B*). Lagging chromosomes and dumbbell shaped nuclei were also occasionally observed (Figure 5*C*). Heteroploid (Figure 5*A*) and diploid cells (Figure 5*D*) were found in the same root, resulting in mixoploids.

Paradichlorbenzene is frequently used

as a household and soil fumigant. It is also one of the constituents of paints applied directly to trees or bushes to control the lilac borer, the flat-headed apple borer and other insects. When used as a soil fumigant, even when applied close to the trunks of trees, there seems to be little danger of producing heteroploidy except, perhaps, in the roots of the plants. However, added care should be taken when the chemical is used in a paint. It should not be allowed to come in contact with the buds because, due to the production of heteroploidy, varietal instability might result.

Summary

1. Roots of *Allium* treated with the vapors of Paradichlorbenzene were found to be stunted.

2. Microscopic examination showed that polyploidy had been produced in these roots.

The possibility that varietal instability may be produced as the result of the use of this chemical in controlling insect pests is suggested.

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EAR PITS AND ALBINISM IN A NEGRO FAMILY

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RECENT authors including Kindred,[2] Whitney,[10] Quelprud,[7] Edmonds and Keeler,[3] Stiles,[9] Connon,[1] and McDonough[4] have shown that ear pit or *Fistula auris congenita* is dependent upon an irregularly dominant gene. Quelprud[7] and Edmonds and Keeler[3] in reviewing the earlier literature upon this subject note that ear pit has been observed since 1864. Stannus[8] found ear pitting in 4.5 per cent of a large group of Nyasaland tribes. Members of pedigrees described in the last few years have all belonged to the white race.

While making a study of albinism in negroes, the authors found one family in which both ear pit and albinism were transmitted by a mother to three of her five children, albinism only to one son, and neither abnormality to one son. A description of albinism in this family, designated the *M* family has been published.[5] In the *M* family, albinism is dominant in its inheritance, having passed through three successive generations in the absence of inbreeding. The albinos of this family appear phenotypically similar to negro albinos of families in which albinism is determined recessively. The pedigree of albinism and ear pits in the *M* family is given in Figure 7. Ear pit is indicated for generations III and IV only since no information was available concerning ear pits in the first two generations. As in previous cases, ear pit is dominant since it is transmitted by the mother, III-3, to three of her five progeny.

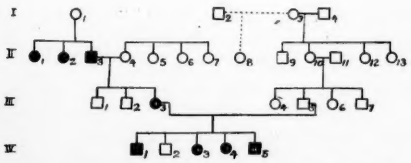
Ear pits in the *M* family occupy the commonest position found in other persons with the abnormality; namely, at the proximal end of the ascending limb of the helix. There existed the usual variability as to presence of ear pit in one or both ears. Photographs of the albino mother, III-3, and her five children are shown in Figure 6. The right ear is placed to the right of the left ear. The mother, III-3, possessed well de-

*The authors wish to thank Dr. Albert Miller of the Department of Tropical Medicine, Tulane University, for assistance in taking the photographs. The authors are also indebted to Dr. P. J. Thomas of Charity Hospital, New Orleans, for assistance in locating the "M" family.

EAR PITS AND ALBINISM IN SIX MEMBERS OF THE *M* FAMILY

Figure 6

The individuals are indicated by symbols referring to the accompanying pedigree chart (Figure 7). That three of the albino children resemble the mother in having ear pits might be due to linkage, but the numbers are too small to constitute more than the suggestion of such a possibility.



PEDIGREE OF ALBINISM AND EAR PITS IN THE *M* FAMILY

Figure 7

Albinism is symbolized by dark circles or squares; the ear pits are indicated by the inner half circles or half squares respectively. The two females (V-3, 4) who have an ear pit only in the right ear are indicated by a single semicircle on the right side.

fined ear pits on both ears. Pits were found on both ears of the youngest albino child, IV-5, and on the right ear only of IV-3 and IV-4. No ear pits were found on the ears of the eldest albino, IV-1, or on his dark brother, IV-2. The photographs shown in Figure 6 were taken in natural sunlight, thus accounting for differences in shade in the two ears. Photographs were unretouched. Owing to an accident, a photograph of the right ear only of the normal dark child, IV-2, was obtained, but it was observed that he lacked ear pits. The normal dark father, III-5, refused to have his ears photographed in detail. It was observed that III-5 also lacked ear pits.

The ear pits were in all cases shallow depressions into which the point of an ordinary lead pencil could be placed. They contained a whitish secretion which caught dirt, giving the appearance of giant blackheads.

Generation IV of the *M* family is also segregating for adherent and free ear lobes. The albino mother, III-3, is seen to possess adherent ear lobes according to Figure 6. Her dark husband, III-5, clearly had free ear lobes according to photographs in the possession of the

authors. Presumably III-5 was heterozygous for adherent lobes since the children IV-1, IV-2, IV-4, and IV-5 have free ear lobes, whereas IV-3 has adherent ear lobes. These data agree with the Powell and Whitney[6] hypothesis that free ear lobe is dominant over adherent ear lobe.

Discussion

There is a suggestion that in this pedigree ear pit may be linked and on the same homolog with dominant albinism in negroes since three of the five children, IV-3, IV-4, and IV-5, inherited both ear pit and albinism from their ear pit albino mother. The father was a normal dark negro without ear pits. One albino son, IV-1, lacked ear pits. He might be regarded as a crossover or he might carry the ear pit gene without showing it phenotypically since it is known that ear pit behaves as an irregularly dominant gene. The normal dark son, IV-2, also lacked ear pits. No conclusion may be drawn at present concerning possible linkage between ear pit and dominant albinism. It is hoped to keep in touch with the *M* family over a period of years.

It is probable that ear pit resulted from independent mutations in the negro and white races. No evidence of white race ancestry was found in the pedigree of the *M* family.

Kindred's[2] case of ear pitting involved the left ear only. Edmonds and Keeler[3] suggest from the data of their two pedigrees that the greater incidence of left ear pits is due to lower metabolic gradients on that side. Whitney[10] also noted that there was a higher frequency of left ear pits. A summary of pit locations in pedigrees described by Edmonds and Keeler,[3] McDonough,[4] Connon,[1] Quelprud,[7] Whitney,[10] and the present authors is given in Table I. The individuals listed in Table I come from pedigrees in which the location of ear pits on one or both ears is variable; that is, Kindred's pedigree is omitted from Table 1. The ratio of left and right ear pits to left ear pits to right ear pits in Table I is 30:28:12. Stannus,[8] on the contrary, found right ear pits with a higher frequency in the African tribes he observed. His ratio is 50 right and left ear pits to 110 left ear pits to 132 right ear pits among a total of 6491 individuals observed. This suggests that no general developmental law favors presence of ear pits on the left side over the right side. More probably lesser modifying genes may affect the developmental process resulting in ear pits differently distributed in various geographic groups.

TABLE I. Summary of ear pit individuals in pedigrees of various authors.

Author	Right and Left	Left	Right
Edmonds and Keeler	6	7	1
McDonough	2	2	1
Connon	0	9	1
Quelprud	17	7	6
Whitney	3	3	1
The Pipkins	2	0	2
Total	30	28	12

Summary

A pedigree has been presented in which dominant albinism and ear pits are inherited in a negro family in such a way as to suggest possible linkage of the two genes. Typical ear pits located at the base of the helix of one or both ears in this negro family resemble such ear pits in white families both in appearance and in dominant type of inheritance.

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COMPLEMENTARY GENES IN WHEAT

Causing Death of F_1 Plants*

E. G. HEYNE, G. A. WIEBE, AND R. H. PAINTER†

CROSSES made to introduce hessian-fly resistance into adapted wheats in Kansas and in California resulted in some hybrids bringing into expression lethal factors in F_1 plants. Marquillo was used in Kansas and Dawson in California as sources of fly resistance. Marquillo was crossed with several soft wheats in Kansas, including Dawson, but all the F_1 plants died before producing grain. Big Club (C.I. 11761)‡ when crossed with Dawson in California, also gave F_1 plants that died in the seedling stages. These lethals were expressed after the plant reached the four or five leaf stage. The seedling emerged normally, but soon after the second leaf appeared the primary leaf began to lose chlorophyll and finally dried up. This disappearance of chlorophyll gradually affected all of the leaves and the plant died. The root system appeared to develop normally for wheat seedlings. Supplemental light and increased temperatures in the greenhouse resulted in more leaves forming, but normal plants were never produced. Figure 8*A* shows the Dawson and Big Club parents and the F_1 plants at maximum development. Figure 8*B* shows the backcross, Oro-Fultz (F_1) × Marquillo.

Lethal F_1 plants in barley have been reported by Wiebe[5] in which the lethal condition was determined by complementary factors, each parent contributing a single dominant factor; and similar genetic explanation has been reported by McMillan,[3] for wheat in Australia, in which the physiological breakdown of the leaves did not occur until flowering time. This "firing" condition was not always lethal, and some affected plants produced grain. Reference to a number of crosses with Marquillo giving lethality in F_1 plants was made by Painter, Jones, Johnston, and Parker.[4] Caldwell[1] reported on the inheritance of a lethal seedling character in a cross between common and durum wheats. A detailed discussion of the lethal necrosis that occurs in some crosses of Marquillo and certain soft wheats has been reported by Caldwell and Compton.[2]

Marquillo has given lethality in F_1 plants when crossed with Fultz, Honor, Shepherd, Dawson, Fulcaster, Harvest Queen, Fulhard, Clarkan, Chiefkan, Red Chief, and Blackhull. Big Club (C.I. 11761) has given lethal F_1 plants when crossed with Dawson and Chiefkan. Marquillo × Big Club (C.I. 11761) gave normal F_1 plants. Both Dawson and Chiefkan gave expression to lethals in F_1 plants when crossed with Marquillo and Big Club (C.I. 11761), which indicates that Marquillo and Big Club have the same factor for lethality. Poso, when crossed with these two varieties, gave normal F_1 plants.

The majority of the soft wheats crossed with Marquillo gave lethal F_1 plants. Michigan Wonder × Marquillo gave normal F_1 plants. Fulcaster and Fulhard apparently are heterozygous for one of the lethal factors, as a number of plants when crossed with Marquillo gave normal F_1 plants. Chiefkan, Red Chief, Blackhull, and Fulhard are the only hard wheats found so far that have given

*Cooperative investigations of the Division of Cereal Crops and Diseases, Bureau of Plant Industry, Agricultural Research Administration, U. S. Dept. of Agriculture, and the Kansas Agricultural Experiment Station, Manhattan, Kans. Contribution No. 348 from the Department of Agronomy and No. 520 Department of Entomology, Kansas Agricultural Experiment Station.

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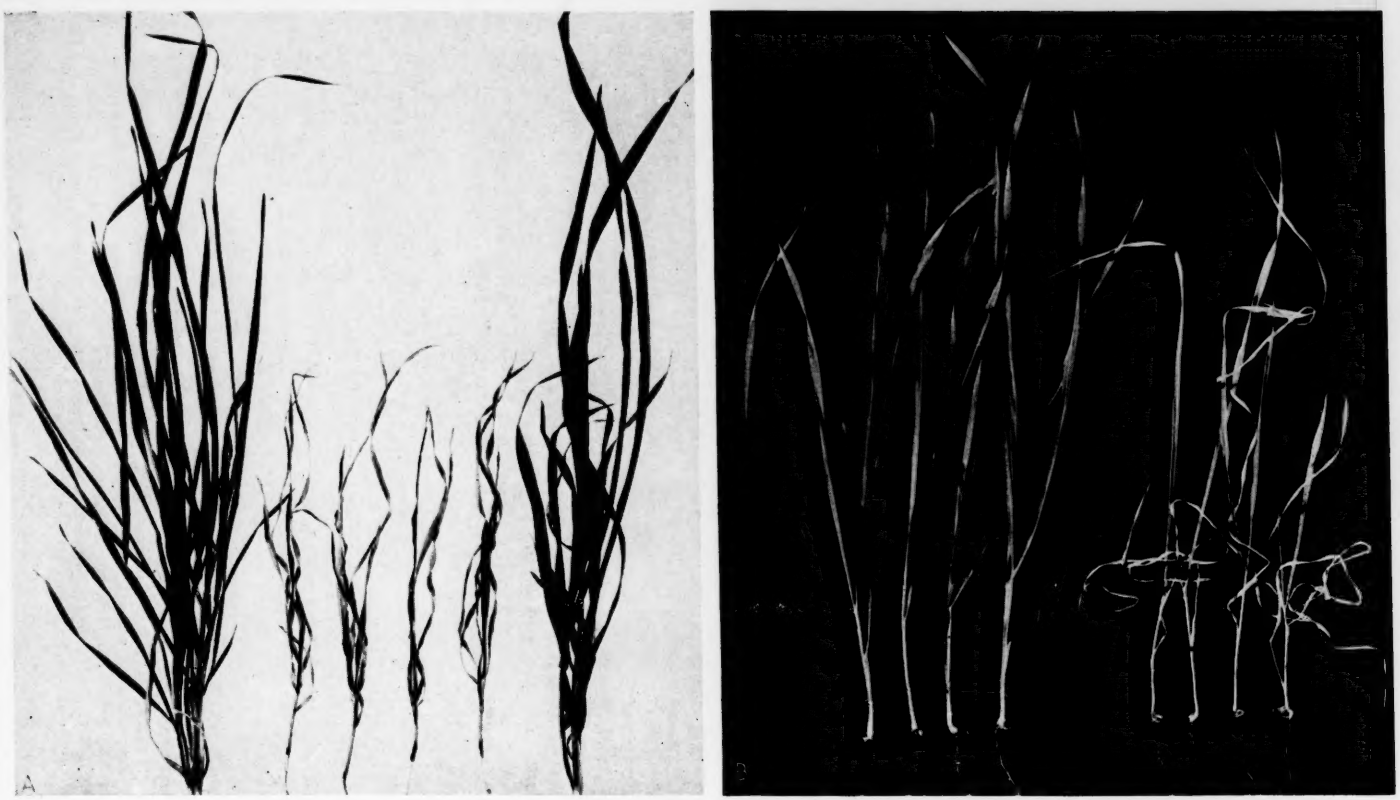
‡C.I. refers to accession number of the Division of Cereal Crops and Diseases.



LETHAL COMPLEMENTARY GENES

Figure 8

A—The plant at the left is Dawson variety, and that at the right is Big Club (CI 11761). The four plants in the center are typical of all the F_1 progency at their maximum development. *B*—shows segregation in the cross Oro-Fultz (F_1) × Marquillo. The four plants at the left are normal and will continue to grow. Those at the right are segregates carrying both of the complementary dominant lethal genes. These plants are destined to die at this stage of growth.

lethal F_1 plants, when crossed with Marquillo.

Data from backcrosses are given in Table I. Marquillo and Big Club (C.I. 11761), each carry one dominant factor, designated as *Le Le,* which in the combination with *Le2 Le2,* carried by the other varieties, gives a lethality in the F_1. Varieties such as Tenmarq, Oro, Cheyenne, Poso, and others carry both recessive *le* and *le2.*

Lethality of the F_1 plants also resulted when the durum wheat, P.I. 94587, was crossed with Thorne or Clarkan. This indicates that Marquillo may have obtained its factor for lethality from Iumillo, its durum parent. This would not give any explanation for Big Club (C.I. 11761), carrying the same factor as Marquillo.

Table I. Backcross data on lethal factors in wheat.

Cross				Segregation	
				Normal plants	Lethal plants
(F_1, Chiefkan x noncarrier*)	x	Marquillo		53	46
(F_1, Clarkan x do	x	do		46	50
(F_1, Dawson x do	x	do		38	51
(F_1, Folcaster x do	x	do		6	7
(F_1, Fulhard x do	x	do		14	16
(F_1, Fultz x do	x	do		21	18
(F_1, Harvest Queen x do	x	do		11	12
(F_1, Shepherd x do	x	do		36	36
			Total	226	237
				expected on basis of 1:1=231.5 : 231.5	
				Range of P = 70 - 80%	
(F_1, Dawson x Big Club, C.I. 11761)	x	Big Club, C.I.11761		92	110
(F_1, Dawson x Poso)	x	do		111	83
(F_1, Big Club, C.I. 11761, x Poso)	x	Dawson		36	43
			Total	239	236
				expected on basis 1:1= 237.5:237.5	
				Range of P = 90 - 95%	

* Noncarrier varieties were Tenmarq, Oro, and Cheyenne.

The data support the conclusion that the lethality in the F_1 of crosses between Marquillo or Big Club (C.I. 11761), and a number of other varieties of wheat is due to dominant complementary factors.

The presence of these genes in wheat may be of use in determining the relationship of varieties. Four wheats, Blackhull, Clarkan, Chiefkan, and Red Chief, developed by Earl G. Clark, former plant breeder in Sedgwick County, Kans., all carry the factor *Le2 Le2,* indicating that these varieties may be related.

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Dr. Crew Appointed "Biological Adviser" to British Army

Biologists generally, and friends of Dr. F. A. E. Crew in particular, will be interested in the following note from the "London Letter" of the *Jour. Amer. Med. Assoc.* for April 17, 1943:

"The role of the medical services as experts in all that concerns the human factor in welfare has been formally recognized by the military authorities. For some time the army council has had a scientific adviser, Sir Charles Darwin, F.R.S., but he and his staff are concerned only with weapons, equipment and ways of making war. A need was felt for a complementary adviser on the biologic side. The director of medical research, Brigadier F. A. E. Crew, F.R.S., will henceforth fulfil this function on the same level as the scientific adviser. In doing so he will require all possible information, on great matters and on small, from the periphery and especially from the field."

THE INHERITANCE OF PLUMAGE COLOR IN THE TURKEY*

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Kansas Agricultural Experiment Station

THE experimental work upon which this paper is based was conducted by Dr. Robertson in the eleven breeding seasons of 1917 to 1927 at the University of Kansas and at the University of Missouri. Following his death early in 1941, the material was dispatched to the Kansas State College Department of Poultry Husbandry where the original records were summarized for publication.

At the inception of this study, no previous critical genetic studies had been reported on the turkey. Only brief reports of this work have been presented by Robertson.[6,7,8,9,10,11] Since that time, however, and previous to the more complete report presented here, other genetic studies have been conducted on the plumage color of the turkey.

Asmundson[1] made crosses involving Bronze and Bourbon Red breeds and found the bronze pattern to be determined by an autosomal factor incompletely dominant to red. He also confirmed Robertson's finding that the Narragansett pattern was inherited as a sex-linked recessive factor (*l*) producing light down color and non-barring of the flight feathers. Still later he[3] demonstrated the presence of an autosomal recessive factor (*sl*) for slate plumage in his turkeys, which shows a reversal of dominance to the factor for slate postulated by Robertson. Therefore, it appears, as pointed out by Asmundson, that the turkey possesses both a dominant and a recessive factor capable of producing the same phenotype.

Another sex-linked recessive factor (*al*) has been described by Hutt and Mueller.[4] It is an imperfect albinism, leaving a faint bronze ghost pattern. The eyes of of such individuals are greatly reduced in pigmentation, the iris being a pale blue grey while the pupil is red, due to the absence of pigment.

Experimental Results

In Robertson's study, data from 88 pair matings were obtained at the University of Kansas and from 469 such matings at the University of Missouri, making a total of 457. A total of 4,800 birds contributed to the color data and 2,633 of these were skinned and the skins preserved. Of the preserved skins slightly less than half were from adult birds, the rest being from poults. Most of the poult skins and many of the adult skins, however, were in very poor condition for accurate description. Consequently it was necessary to rely upon the descriptions supplied with the data, and unless otherwise noted all such descriptions are those of Dr. Robertson. In most cases it was not possible to determine whether down, juvenile, or adult plumage descriptions were involved and whenever it appeared that Dr. Robertson was in doubt as to the genotypes of the offspring, the results were not included in the summary. In addition to the original descriptions of the offspring of the various matings, there were avail-

*Contribution No. 148 Department of Poultry Husbandry.

†Deceased before preparation of this manuscript, while a member of the staff of the Medical School of the University of Iowa. The authors preparing this paper are indebted to the Department of Anatomy of the University of Iowa for making available to them for study the materials and data collected by Dr. Robertson and to the Missouri Agricultural Experiment Station and the Department of Zoology of the University of Kansas where the data were collected.

‡Now at Purdue University, Department of Poultry Husbandry.

able fragmentary preliminary drafts of manuscripts by Robertson.

Robertson used but very few purebred birds and when doing so mated them promiscuously to all types of crossbreds. Such a procedure would ordinarily have resulted in data of virtually no value. In the case of turkey colors the possibility of recognizing most factors in varying combinations made it feasible to analyze such material genetically. Therefore, most of the matings involved more than one factor pair difference, which made the data difficult to analyze. In order to minimize this difficulty, all matings were studied in relation to only one factor at a time. Consequently birds homozygous for a given factor pair might have been heterozygous for some other factor not under consideration. In other words, birds considered to be the P_1 generation were necessarily homozygous for only the factor under study while no consideration was given to the bird's constitution with respect to other factors unless they tended to mask the factor under consideration. While such a procedure could introduce errors of description due to epistasis, such an effect is infrequent, as is indicated by the good fit of the actual progeny numbers to the expected ratios. Birds whose genotypes were difficult to classify by external pattern were analyzed in test crosses by Robertson to obtain the desired information.

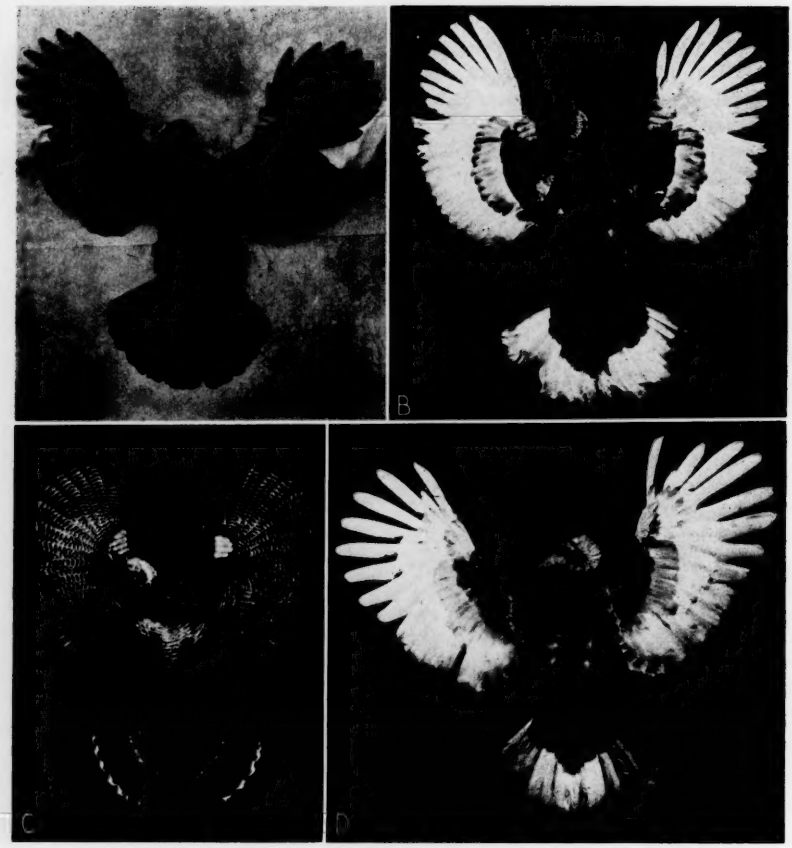
The summarization of the data, a factor pair at a time, had the effect of materially expanding the number of individuals in any given cross. This system of analysis utilized by the authors preparing this manuscript was not followed by Robertson since he appeared to have attempted to deal simultaneously with all factors involved in any pair mating. The confusion encountered in such a procedure may have contributed to the long delay in the presentation of the original data. Nevertheless, the fact that similar conclusions were reached by both procedures is added evidence of their validity.

Black × Bronze Matings

Of the domestic varieties of turkeys, the Bronze (Figure 9*C*) most nearly approaches the wild type in color pattern. In fact, Robertson's data show definitely that the genetic composition of the two is identical with respect to major color factors. In this pattern the contour feathers of the female are black with a wide bronze band near the end, followed by a narrow distinct black band and finally terminating in a white edging, the width of which increases on each feather toward the posterior part of the body. The body of the male is similar to the female in marking except that the feathers lack the white tips. The main tail and covert feathers of both sexes are marked with alternating black and chestnut red bars. Each of these feathers terminates in a white band, immediately proximal to which is a wide bronze bar distinctly bordered with intense black. The primary and secondary flight feathers are marked with alternating black and white bars of approximately equal width. The eyes are dark brown according to the American Standard of Perfection, while Robertson describes the eyes of his birds as brilliant bay-red.

The entire plumage of the Black variety is black with a bright metallic sheen, preferably showing no evidence of bronze, white or red barring, or white tips (Figure 9*A*). Robertson describes the iris of the eye to be black in contrast to the bay-red of the Bronze whereas the standard description calls for a dark brown eye as in the cast of the Bronze. Poults of this variety have slaty blue shanks which gradually fade out to the adult pink shade.

Examination of Table I reveals that reciprocal crosses between Black (*B*) and Bronze (*b*) always gave most entirely black offspring of both sexes. This indicates that the factor difference between these two patterns is autosomal and that black is almost completely dominant. The F_1 "black-bronze" (*B*) individuals were black except for a few bronze or partially bronze feathers scattered through the plumage. In Figure



COLOR PATTERNS IN THE TURKEY
Figure 9

A—Standard Black ♀ (*CCBBRRddN*—); *B*—Bourbon Red ♀ (*CCbbrrddN*—); *C*—Standard Bronze ♂ (*CCbbRRddNN*); *D*—Buff ♀ (*CCBBrrddN*—).

10*A* several such feathers are seen among the tail coverts.

The F_2 generation from these crosses consisted of 93 black (*BB*), 184 black-bronze (*Bb*), and 75 bronze (*bb*) individuals, the numbers closely approximating the expected 1:2:1 ratio. A X^2 value of 2.55, with $p = 30$, attests to the goodness of fit.

Reciprocal backcrosses of the heterozygotes (*Bb*) to black (*BB*) gave 22 black (*BB*) and 26 black-bronze (*Bb*) individuals or approximately equal proportions ($X^2 = 0.33$; $p = 50+$). Similarly, reciprocal backcrosses of the heterozygotes (*Bb*) to bronze* (*bb*) gave 302 bronze (*bb*) and 306 black-bronze (*Bb*), again appearing in equal propor-

*Throughout this paper capital letters have been used only when reference is made to a named variety while lower case has been used to designate the phenotypic expression of the various pattern factors.

tions ($X^2 = 0.026$; $p = 50+$). These data clearly indicate a one-factor difference between the color patterns of the Black and Bronze varieties.

It was further noted by Robertson that when the gene *B* for black plumage was present, the eyes were also black, whereas if the gene *b* for bronze plumage was present, the eyes were bay-red.

Bronze × Red Matings

The red pattern of the Bourbon Red variety differs from the bronze in the almost complete absence of black, the only traces being a few scattered granules in the region where the broad black subterminal bars would appear in the bronze pattern. In addition the males have a narrow black terminal edging to the feathers of the breast and anterior parts of the body (Figure 9*B*). Although lacking the black edging on the feathers characteristic of the males, the breast feathers of the female are marked with a very narrow edging of white. The remainder of these tipped feathers and other body feathers in both sexes are a rich chestnut red. The primary, secondary, and main tail feathers are pure white except for a dim soft red sub-terminal bar crossing each tail feather. The eyes are dark brown as in the Bronze and the shanks are a light pinkish red.

Reciprocal crosses between Bronze (*RR*) and Red (*rr*) resulted in an F_1 of intermediate color, the bronze-red (*Rr*) (Table II). The intermediates (Figure 8*C*) were more like the bronze than the red, but clearly showed the influence of the *r* factor by the decrease in width of the black bars with a corresponding increase of the chestnut red, giving the bird a general reddish appearance. Another distinctive characteristic of the heterozygote was the partial breaking up of the black bars in the flight feathers with an accompanying increase in the amount of white and a sprinkling of red.

Individuals of the F_2 generation segregated into 52 bronze (*RR*), 124 bronze-red (*Rr*) and 66 red (*rr*), the 1:2:1 ratio being closely approximated ($X^2 = 1.76$; $p = 40+$). Backcrosses of heterozygotes to bronze (*RR*) gave 92 bronze and 108 bronze-reds which is approximately equal proportions ($X^2 = 1.28$; $p = 20+$), while backcrossing to the red (*rr*) gave 76 bronze-reds and 57 reds, approximating the 1:1 ratio ($X^2 = 2.7$; $p = 10$).

In Table II are shown the results if red is considered as the allelomorph of non-red or the extension of black. Non-red thus consisted of the bronze, black, slate or narragansett patterns. None of the results differ significently from the expected ratios (F_2, $X^2 = 2.55$; $p = 20+$: backcrosses to non-red, $X^2 = 2.67$; $p = 10$: backcrosses to red, $X^2 = 2.66$; $p = 10$). Thus the factor *R* in the turkey might be considered as having an effect similar to that of the extension factor *E* in the fowl. Again the data indicate that a one factor difference exists; this time between the bronze and the red breeds, or even between red and non-red.

Black × Red Matings

As in the previous cross, matings between Black (*BBRR*) and Red (*bbrr*) gave an intermediate color, black-red (Table IV). Such black-red intermediates might be called rusty blacks for they show a considerable increase of red particularly in such posterior feather tracts as the main tail, tail coverts and some in the saddle (Figure 9*E*). This red color takes the form of an irregular penciling, varying toward a type of heavy stippling. A distinct tendency for white barring to appear in the wings has also been observed. Backcrossing the heterozygotes to the black parent resulted in 28 black, 31 black-red, and 15 black-bronze individuals, a proportion which though skewed toward the black is not significantly different from the 1:2:1 ratio ($X^2 = 5.34$; $p = 7$). The reason for this ratio in a backcross was that the *BB* individuals could not be segregated accurately from those carrying *Bb* in the presence of *Rr*. Backcrosses to the red parent gave equal classes of four types, namely, 30 black-red intermediates, 32 bronze-red intermediates,

25 pure reds, and 22 of a new type, buff ($X^2 = 2.30$; $p = 50$). The new color, buff, is similar to the bourbon red except for a marked reduction in the intensity of the red pigment, very dark eyes like the black breed and chocolate colored shanks (Figure 8*D*). Such birds were demonstrated to carry the black gene (*B*) which apparently tended to reduce the intensity of the red pigment, a reaction totally unexpected in the light of its effect in the presence of *R*. The F_2 generation segregated as 13 black, 17 black-bronze, 65 black-red, 31 buff, 12 bronze, 11 bronze-red, and 10 red. This ratio is close to expected ($X^2 = 6.25$; $p = 30$) on the basis of a two factor difference where, in two cases, classes could not be distinguished phenotypically. Here again, birds homozygous for black (*BB*) could not be distinguished from the heterozygotes (*Bb*) in the presence of *Rr*; nor could they be identified accurately in the presence of *rr* or buff birds. These results indicate that two factor pairs are involved in the black × red cross. It follows then, that the factor difference between the bronze and black patterns is different than that between the bronze and red patterns.

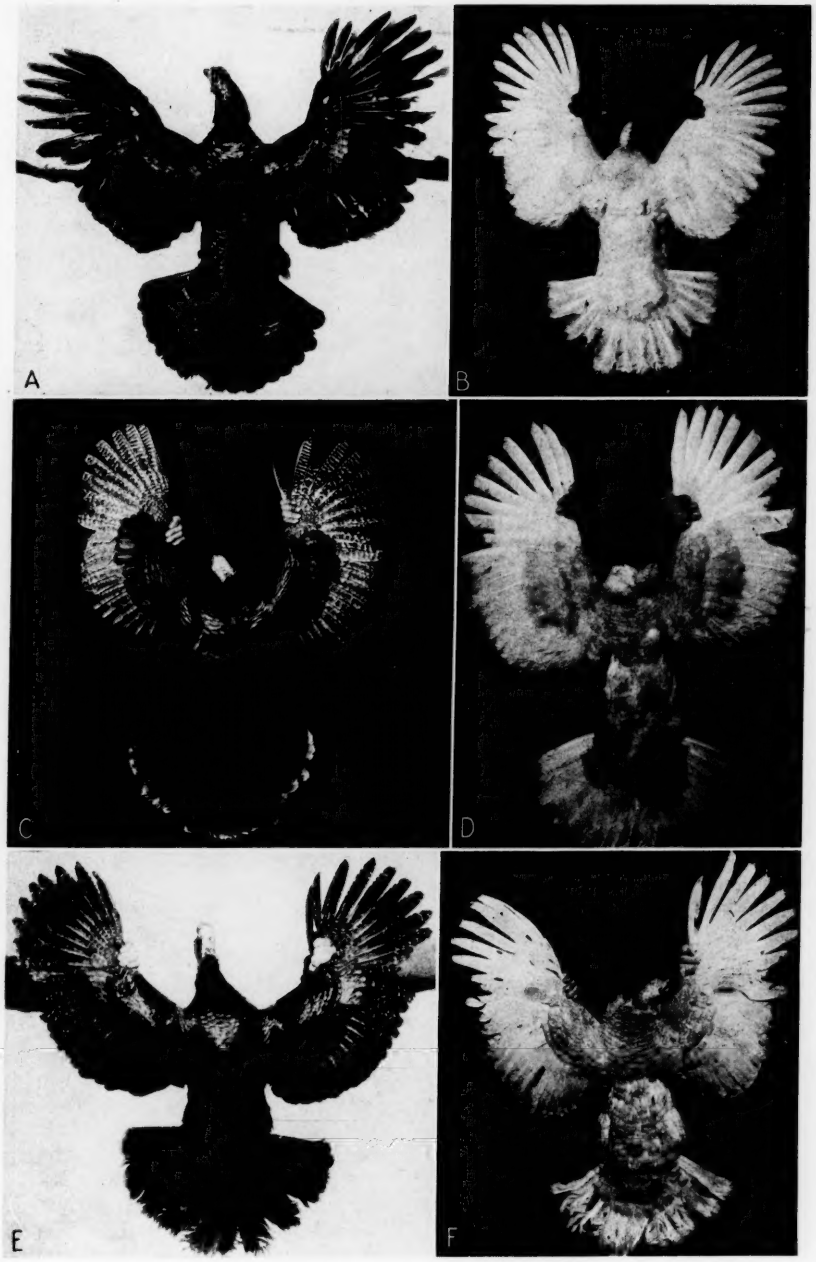
Inheritance of Slate

Robertson[11] originally used the symbol *Sl* to represent the dominant slate gene. Since that time, however, Asmundson[3] has applied the symbol *sl* to the recessive factor for slate. Consequently it seems advisable to adopt the symbol *D* as suggested by Marsden and Martin [5] for the dominant slate determiner in order to avoid confusing the two non-allelomorphic factors.

A uniform slaty blue color is desired in the Slate variety (Figure 9*D*). The slate gene *D* reduces the black pigment of any plumage pattern to a slaty blue but has little or no effect on red pigment. Therefore, the slate gene could be identified only in patterns showing definite black pigment. Reciprocal crosses of slate (*DD*) and non-slate (*dd*) gave all slate offspring (*Dd*) (Table V), as did the backcrosses to the slate parent. Backcrosses to non-slate, however, approached a 1:1 ratio with 299 slate and 327 non-slate offspring ($X^2 = 1.25$; $p = 20$). It was very difficult to separate homozygous dominant slates from heterozygotes, an indication, though not entirely reliable, being the black flecking in the blue feathers of the heterozygote (Figures 9*F* and 10*B*). Consequently the slates were all combined in the F_2 generation resulting in a near approach to a 3:1 ratio of slate to non-slate ($X^2 = 2.39$; $p = 10$). Test crosses were frequently employed to determine parental genotypes where this factor was involved.

The Slate variety is somewhat irregular in breeding, a common undesired type being a rather dark slate with frequent off-color markings tending to show the color pattern of the bronze (Figure 10*A*). The bronze pattern is distinctly noticeable particularly in the barred wing feathers, and the failure of the slate gene to dilute the red pigment of the tail and its coverts allows this chestnut red to stand out even more definitely against the slate background than it does in the black feathers of the bronze.

Robertson's data indicate that the gene for black (*B*) in combination with the dominant slate (*D*) gave a uniform bluish slate color such as desired in the standard-bred Slate variety. This color phase has been called "blue" in Robertson's early reports. The uniformity of color would be expected since the gene *B* would give all of the plumage a black color which in turn could be diluted through the action of the slate gene *D*. For the second time an unexpected action of the black gene *B* was evident. The first time such action was mentioned was when it was combined with red (*rr*) to produce buff, while in this case it interacts with slate (*D*) to produce the lighter shade. Although many crosses were involved in establishing this point, none could be considered as entirely critical. The nearest approach to such a cross was between light slate birds heterozygous for slate and homozygous for black (*Dd BB*). The offspring produced were 10 light slates and 4 blacks. Since



COLOR PATTERNS IN THE TURKEY

Figure 10

A—Black-bronze hybrid ♀ (*CCBbRRddN*—); *B*—White Holland ♂ (*ccbbRRddNN*); *C*—Bronze-red hybrid ♂ (*CCbbRrddNN*); D—Standard (Light) Slate ♀ (*CCBBRRDDN*—); *E*—Black-red hybrid ♂ (*CCBbRrddNN*); *F*—Heterogygous Light Slate ♀ (*CCBBRR-DdN*—).

only light slate and black offspring in a 3:1 proportion were expected from such a cross, the results are very close to those expected on the basis of the hypothesis ($X^2 = 0.088$; $p = 50^+$). Since standard slates might be heterozygous for black, they did not necessarily breed true. Birds carrying bronze (*bb*) together with the factor for slate (*D*) showed a uniform dilution of the black pigment but exhibited little or no effect on the red and white markings, leaving a faint bronze pattern with considerable red in the tail region. Such birds, if homozygous for slate should breed true for this color phase.

Taibel[12] crossed two slate turkeys and obtained seven slate and five bronze offspring as well as four uniformly colored light slates which were called "lilacs." His conclusion on the basis of these small numbers was that slate was a heterozygous condition much as the blue color in chickens. It is difficult to determine wherein lies the discrepancy between these data and those of Robertson, but the small numbers reported by Taibel make the genetic analysis of the factors involved inconclusive.

Asmundson[3] reported a recessive factor for slate to which he applied the symbol *sl*. Here again two color phases occur. He presented evidence that the darker type with barred flight feathers and penciled tail feathers also carried the gene *R* for extended black pigment. Such birds do not show so much red in the tail region as do the dominant slates. The lighter colored individuals with almost clear white flight feathers and non-penciled tail feathers, which he also referred to as "lilac," carried the gene *r* for the bourbon red pattern in the homozygous condition.

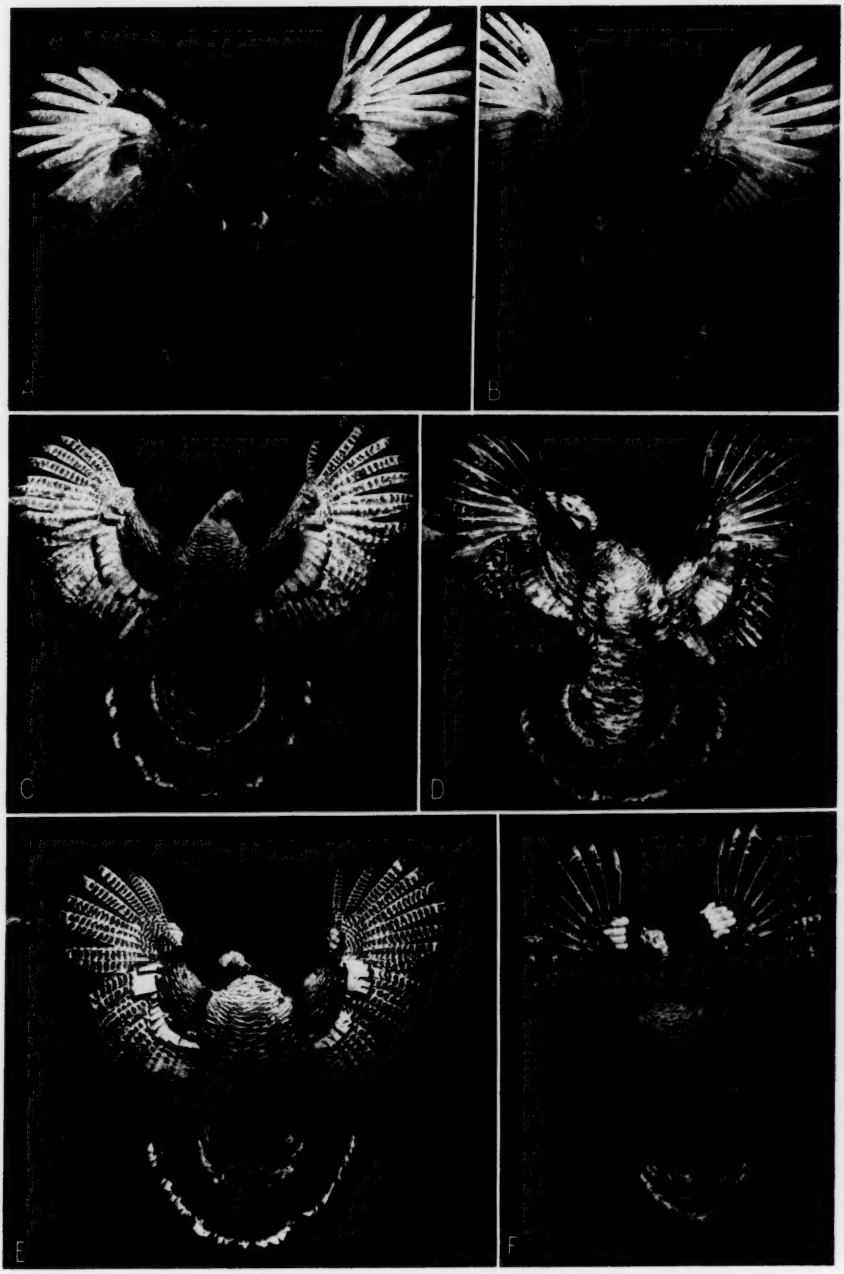
Inheritance of the Narragansett Pattern

The Narragansett variety has the same general pattern as the bronze except for a graying effect in the subterminal bronze bands giving the bird a silvery appearance (Figure 10*E*). This graying is so extreme on the neck that the entire exposed surface is gray except for a narrow black edging, while the unexposed parts of the feathers in this region are black. The same pattern carries to the wing front and bow, and to the secondary coverts. The latter have a large exposed silvery area forming a characteristic wide silver bar across the folded wing, while the black tips of these feathers form a border of black. The black progressively encroaches on the silver-gray toward the posterior part of the body until the back feathers are a rich black with no traces of silver and preferably no bronzing effect. The main tail feathers and tail coverts are black, regularly penciled with tan. Each feather has a broad band of metallic black near the end and a wide edging of silver. The primaries, secondaries, and primary coverts are marked with regular black and white bars. The female has an additional white edging on all body feathers, which increases in width toward the rear of the bird. The eyes are brown and the shanks are a pinkish brown.

When narragansett females (*n*-) were mated with non-narragansett males (*NN*), all of the offspring were non-narragansett, indicating that the determiner for this factor is a recessive (Table VI). The reciprocal cross using narragansett sires (*nn*) gave all narragansett females and all non-narragansett males where the sex was obtained, suggesting a sex-linked mode of inheritance.

Matings of F_1 non-narragansett males (*Nn*) to non-narragansett F_1 females (*N*-) resulted in 172 males and 86 females of the non-narragansett pattern and 86 females showing the effect of the narragansett factor. Since none of the males and half of the females would be expected to show the narragansett pattern on the basis of a sex-linked recessive gene, these experimental results are a perfect fit to expected numbers.

Crossing similar F_1 males (*Nn*) with the narragansett pattern F_1 females (*n*-) produced males and females of both color types. A highly significant shortage of narragansett offspring ($X^2 = 8.56$; $p = < 1$) might be due to the difficulty of positive identification of this factor in combination with red. However, in view



COLOR PATTERNS IN THE TURKEY

Figure 11

A—Dark Slate ♂ (*CCbbRRDDNN*); *B*—Heterozygous Dark Slate ♀ (*CCbbRRDdN—*); *C*—Narragansett-red hybrid ♀ (*CCbbRrddn—*); *D*—Gray ♀ (*CCBbRrddn—*); *E*—Standard Narragansett ♀ (*CCbbRRddn—*); *F*—Silver Tipped Black ♀ (*CCBBRRddn—*).

of the absolute relation of sex to color pattern, this discrepancy is insufficient to discredit the hypothesis of the sex-linked recessive nature of the narragansett factor.

Inheritance of White

Very few matings were made to study the inheritance of white (Figure 9*B*). Only colored offspring were obtained from reciprocal matings of white (*cc*) and colored (*CC*) birds, but no records were obtained as to the sex of the poults (Table VII). The 15 white and 13 colored offspring from backcrosses to white were of both sexes. The F_2 generation consisted of 23 colored to 7 white individuals, a proportion very close to the expected 3:1 ratio ($X^2 = 0.044$; $p = 50^+$). These data indicate the action of an autosomal recessive determiner for white plumage.

Effect of Five Plumage Color Factors in Combination

The dominant slate gene as observed by Robertson may be combined with any other plumage color genes studied. Thus, in combination with black, it produces the light shade of slate previously described (Figure 9*D*). With black-red it produces a cinnamon-slate (*Sl-BbRr*). Such individuals show the same markings as the F_1 generation from the black-red cross but the black areas are of the standard light slate while the red or white markinigs remain uninfluenced. Combined with ordinary bronze it produces a darker slate (Figure 10*A*) whereas with bronze-reds it produces a reddish slate having the typical markings of the F_1 intermediate from the bronze-red cross. The gene has no effect on the red pigment of the buff or red patterns but does reduce the black edgings on the feathers of the males to a slate color. Such changes in reds or buffs were difficult to recognize, however, since so small an area was affected.

The narragansett factor (*n*) changes the bronze color patterns to that characteristic of the Narragansett variety (Figure 3*R*). When combined with the gene *B* for black it produces a color pattern which may be described as silver-tipped black (Figure 10*F*). These birds do not show the extreme graying effect on the neck, wing bows, and wing bars. The feathers of the anterior parts of the body exhibit only a clear black color. The main tail, tail coverts, and saddle feathers all have a wide edging of the silver gray, but no evidence of red pigment is seen in any region. The flight feathers further show distinct traces of white barring. In combination with black-red heterozygotes the pattern is the same as in combination with black except that considerable light red appears in the tail coverts, saddle, and main tail feathers, and the silvering effect is very strong in the anterior feather tracts. Such specimens are best described as gray (Figure 10*D*). Combined with bronze-red heerozygotes it produces a reddish narragansett pattern.

This narragansett factor appears to be the only gene other than black (*B*) which is capable of reducing the intensity of red pigment. Thus when the genes for narragansett (*n*) and black (*B*) are combined with red (*rr*) a very light buff is produced. The narragansett gene by itself reduces markedly the intensity of the red pigment of the Bourbon Red pattern. Another example of this effect on red pigment may be observed by contrasting the tan penciling of the Narragansett variety with the chestnut red penciling of the Bronze.

The ordinary White Holland variety of turkeys in all birds tested carried the bronze pattern as a cryptomere. However, white (*cc*) is also epistatic to all of the other color patterns studied, as specimens with any combination of color factors were white as long as they carried the recessive *c* in a homozygous condition. The genetic composition of white specimens was readily determined by outcrossing to colored birds of known genotypes.

Table VIII contains a list of all recognizable phenotypes which might result from combinations of the five factors discussed in this paper, accompanied by the possible corresponding genotypes. Female genotypes were omitted from

Table I. Black x Bronze Matings

Parents	Offspring						
	Type		♂	♀	Sex ?	Total	Expected
Black ♂ x Bronze ♀	Black-bronze	Bb	36	68	64	168	168
Bronze ♂ x Black ♀	Black-bronze	Bb	31	63	58	152	152
F_2	Black	BB	31	40	13	93	88
	Black-bronze	Bb	73	81	30	184	176
	Bronze	bb	38	28	9	75	88
Backcrosses to Black	Black	BB	8	14	--	22	24
	Black-bronze	Bb	9	17	--	26	24
Backcrosses to Bronze	Black-bronze	Bb	125	137	44	306	304
	Bronze	bb	110	147	45	302	304

Table II. Bronze x Red Matings

Parents	Offspring						
	Type		♂	♀	Sex ?	Total	Expected
Bronze ♂ x Red ♀	Bronze-red	Rr	53	50	47	150	150
Red ♂ x Bronze ♀	Bronze-red	Rr	91	106	42	239	239
F_2	Bronze	RR	14	20	18	52	60.5
	Bronze-red	Rr	41	41	42	124	121
	Red	rr	28	18	22	68	60.5
Backcrosses to Bronze	Bronze	RR	34	32	26	92	100
	Bronze-red	Rr	42	39	27	108	100
Backcrosses to Red	Bronze-red	Rr	32	30	14	76	66.7
	Red	rr	17	29	11	57	66.7

Table III. Red x Non-red Matings

Parents	Offspring						
	Type		♂	♀	Sex ?	Total	Expected
Red ♂ x Non-red ♀	Intermediate	Rr	77	84	55	216	216
Non-red ♂ x Red ♀	Intermediate	Rr	130	135	63	328	308
F_2	Non-red	RR	35	47	23	105	120
	Intermediate	Rr	99	104	49	252	240
	Red	rr	48	47	28	123	120
Backcrosses to non-red	Non-red	RR	170	170	80	340	362
	Intermediate	Rr	109	161	57	384	362
Backcrosses to red	Intermediate	Rr	58	72	19	147	138.5
	Red	rr	35	55	20	110	138.5

Table IV. Black x Red Matings

Parents	Offspring						
	Type		♂	♀	Sex ?	Total	Expected
Black ♂ x Red ♀	Black-red	BbRr	--	--	5	5	5
Red ♂ x Black ♀	Black-red	BbRr	20	20	10	50	50
F_2	Black	BBRR	4	8	1	13	10
	Black-bronze	BbRR	9	8	--	17	20
	Black-red	BbRr	26	26	5	66	60
	Buff	Bbrr	9	18	4	31	30
	Bronze	bbRR	5	5	2	12	10
	Bronze-red	bbRr	8	3	--	11	20
	Red	bbrr	5	4	1	10	10
Backcrosses to Black	Black	BBRR	8	9	11	28	18.5
	Black-red	BbRr*	5	12	14	31	37
	Black-bronze	BbRR	4	3	6	13	18.5
Backcrosses to Red	Black-red	BbRr	10	18	2	30	27.25
	Buff	Bbrr	5	12	6	22	27.25
	Bronze-red	bbRr	11	18	3	32	27.25
	Red	bbrr	10	12	3	25	27.25

* RR individuals could not be segregated from those carrying Rr in the presence of Rr or rr.

Table V. Slate x Non-slate Matings

Parents	Offspring						
	Type		♂	♀	Sex ?	Total	Expected
Slate ♂ x non-slate ♀	Slate	Dd	31	34	5	70	70
Non-slate ♂ x Slate ♀	Slate	Dd	3	9	1	13	13
F_2	Slate	Dd or DD	121	140	18	279	293
	Non-slate	dd	48	55	8	111	98
Backcrosses to slate	Slate	Dd or DD	14	15	1	30	30
Backcrosses to non-slate	Slate	Dd	141	133	25	299	313
	Non-slate	dd	127	138	32	327	313

Table VI. Narragansett x Non-narragansett

Parents	Offspring						
	Type		♂	♀	Sex ?	Total	Expected
1. Narr. ♂ x Non-narr. ♀	Non-narragansett	Nn	100	--	6	106	110
	Narragansett	nn	--	112	3	115	110
2. Non-narr. ♂ x Narr. ♀	Non-narragansett	Nn or N-	78	82	69	229	229
3. F_2 from mating 1	Non-narragansett	Nn or N-	66	69	--	135	113
	Narragansett	nn or n-	41	51	--	92	113
4. F_2 from mating 2	Non-narragansett	Nn or N-	172	86	22	280	279
	Narragansett	nn	--	86	5	91	92

Table VII. White x Colored Matings

Parents	Offspring						
	Type		♂	♀	Sex ?	Total	Expected
White ♂ x Colored ♀	Colored	Cc	--	--	18	18	18
Colored ♂ x White ♀	Colored	Cc	--	--	18	18	18
Backcrosses to white	Colored	Cc	1	4	8	13	14
	White	cc	5	3	7	15	14
F_2	Colored	CC	9	12	2	23	22.5
		Cc					
	White	cc	3	4	--	7	7.5

Table VIII. Action of Five Plumage Color Factors in Combination in Male Turkeys

Phenotype	Genotype
Standard slate	N?B?RrD?C?*
Standard black	N?BBRrDdC?
Slate with much red in back and tail	N?B?RrD?C?
Smoky black	N?B?rrddC?
Slate tipped buff	N?B?rrD?C?
Buff	N?B?rrddC?
Black with few scattered bronze feathers	N?bbRRDdC?
Dark slate with much red in back and tail	N?bbRrD?C?
Dark slate, faint bronze pattern	N?bbRrDdC?
Standard bronze	N?bbRRddC?
Bronze-red intermediate	N?bbRrddC?
Slate tipped red	N?bbrrD?C?
Standard bourbon red	N?bbrrddC?
Light slate	nnB?RrD?C?
Silver tipped black	nnB?RRDdC?
Light slate with light red in back and tail	nnB?RrD?C?
Grey	nnB?RrddC?
Slate tipped light buff	nnB?rrD?C?
Light buff	nnB?rrddC?
Light slate, faint bronze pattern	nnbbRrD?C?
Light slate, faint bronze pattern with light red in tail	nnbbRrD?C?
Standard narragansett	nnbbRRddC?
Reddish narragansett	nnbbRrddC?
Slate tipped, light red	nnbbrrD?C?
Light red	nnbbrrddC?
White	Any combination of pattern genes with cc

* Question marks indicate that either the dominant or recessive allel may be present.

Table IX. Typical Cross Involving Several Factors

Parents	Offspring			
	Type		♂	♀
♂ Narragansett (nnddbbRR) X ♀ Slate black red (B-DdBbRr)	Narragansett slate black red	(B-DdBbRr)		3
	Narragansett slate black	(B-DdBbRR)		4
	Narragansett slate bronze red	(B-DdbbRr)		1
	Narragansett slate	(B-DdbbRR)		4
	Narragansett black red	(B-ddBbRr)		4
	Narragansett black bronze	(B-ddBbRR)		9
	Narragansett bronze red	(B-ddbbRr)		2
	Narragansett	(B-ddbbRR)		4
	Slate black red	(bbDdBbRr)	6	
	Slate black bronze	(bbDdBbRR)	4	
	Slate bronze red	(bbDdbbRr)	1	
	Slate	(bbDdbbRR)	0	
	Black red	(bbddBbRr)	7	
	Black	(bbddBbRR)	1	
	Bronze red	(bbddbbRr)	0	
	Bronze	(bbddbbRR)	1	

this table since they would be the same as the male except that where the narragansett pattern appeared in such females, the necessary factor would be present in a hemizygous recessive condition rather than being homozygous.

As stated previously, most of the crosses made by Robertson involved more than one factor pair. Table IX shows the analysis of an original mating. This particular mating was chosen as an illustration since all of the color factors were involved except that for white plumage. Other matings differed in from one to all five factor pairs under consideration. It is evident that it would be difficult to produce enough offspring from a few single pair matings to obtain accurate genetic ratios. Nevertheless, the fact that the characters segregated so accurately into nearly all expected classes and that no unexpected phenotypes appeared, is still further evidence favoring the hypothesis advanced by Robertson.

Summary

This paper presents a summary of the data collected by the late Dr. W. R. B. Robertson on the genetic factors controlling the color differences among the common varieties of the domestic turkey. These data indicate that black differs from the bronze pattern by an almost completely dominant autosomal factor (*B*), the heterozygotes showing a few scattered bronze or partly bronze feathers in their otherwise black plumage. Red differs from bronze by an incompletely dominant autosomal factor (*r*). Heterozygotes in this case show an increase in the amount of red pigment in an otherwise bronze pattern. This intermediate type is readily distinguishable from either of the parent types. Evidence is presented in F_2 and backcross matings that these are distinct genes and not allelomorphs.

The slate factor *D* is dominant to nonslate. When interacting with the gene for black *B* it results in a uniform slate color. Combined with the bronze factor *b*, it produces the dark slate with a faint bronze pattern and some red pigment showing in the tail region. This gene has no effect on the red pigment of the buff or red patterns but does reduce the black edgings on the body feathers of the males to a slate color.

The factor for the narragansett pattern *n* behaves as a sex-linked recessive. In combinations with the bronze factor *b* it produces the pattern characteristic of the Narragansett variety. With the gene for black it produces a silver tipped black. Combined with the red or buff patterns it serves to reduce the intensity of the red pigment producing light red and very light buff individuals.

White, the absence of pigment, is inherited as a simple autosomal recessive *c* which is epistatic to all factors for color. Although specimens of the White Holland variety in all cases tested carried the bronze pattern gene as a cryptomere, any combination of the color factors could be present in a white bird but would be unable to express themselves phenotypically.

It is to be emphasized that the significant facts regarding color inheritance in turkeys were presented earlier in abstract form by Robertson (1926, 1929) and that this independent summarization of the data by the junior authors seems to substantiate fully Robertson's early deductions.

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